doctors who are not specialist cardiologists are unaware of the existence of this syndrome. This is partly because of the paucity of published reports which draw attention to its presentation as epilepsy.[6]

The proper treatment for the Romano-Ward syndrome is unknown. The variety of methods of treatment suggests that most are only partially effective. Early reports of less frequent convulsions during treatment with beta-blockers have proved disappointing.[7] Most other antiarrhythmic agents have been used, as has left stellate ganglionectomy.[8-10] At present we have treated our patient with phenytoin 300 mg daily, which shortened the Q-T_c interval to 0·49 seconds (fig 3). As suggested[11] we intend to assess the threshold for arrhythmias by autonomic stimulation to determine if this is satisfactory treatment.[7,12]

Unexpected death in a young epileptic, not resulting from status epilepticus or concomitant injury during a fit, is a rare event. It is unknown how many such deaths result from cardiac disorders. In a retrospective analysis of 19 deaths over 20 years in Cleveland, of the few patients who had electrocardiograms recorded, only one had prolongation of the Q-T interval.[13] The allied syndrome of Jervell and Lange-Nielsen, where congenital deafness is associated with lengthening of the Q-T interval, is clearly more easily recognised than is the Romano-Ward syndrome. The results of a survey of deaf children showed the prevalence of the former to be as high as 1%.[14] We suspect that the Romano-Ward syndrome may not be as rare as it appears but that it is frequently unrecognised.

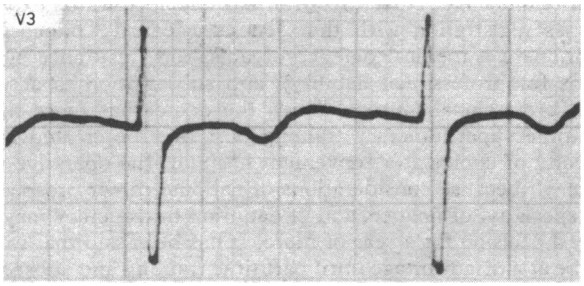

FIG 3—Shortened Q-T interval of the patient in case 1 who was treated with phenytoin 300 mg daily.

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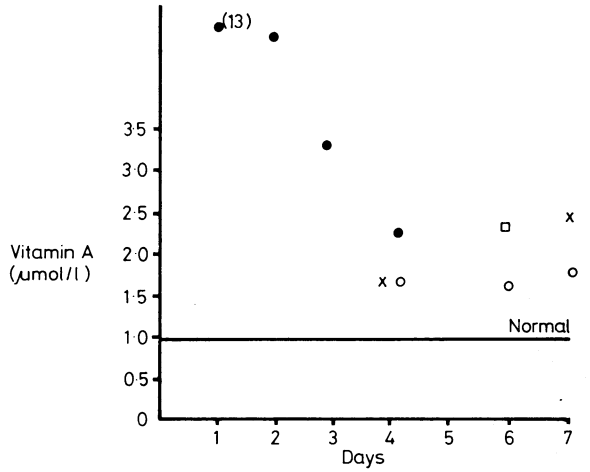
Clinical curio: acute vitamin A toxicity from ingesting fish liver curry

Eleven patients (five men, six women) aged from 15 to 45 years were studied at the medical unit of the General Hospital, Colombo, Sri Lanka. The patients belonged to three different families and were taken ill after a meal that included the liver curry of the fish *Istiophorus glàdius* (sailfish, Sinhala: Talapath). The quantity of fish eaten was about 113 g (4 oz) each. All patients complained of headache and drowsiness, the headache being severe and constant occurring within five to eight hours of the meal; one patient experienced nausea and five vomiting; blurring of vision and redness of eyes without diplopia was present in six cases; and a superficial peeling dermatitis was noted in nine of the patients in the face, neck, and back of chest; it appeared 48 to 72 hours after the meal and lasted from two to four days and regressed without specific treatment. Vitamin A concentrations were measured in four patients (in three of them temporal measurements were made) by a standard method using hexane extraction and using β carotene concentrations (figure).[2] All values obtained were higher than the normal for an age matched population (0·7-0·9 μmol/l (20-27 μg/100 ml), mean 0·8 μmol/l (24·7 μg/100 ml)). Highest values were noted in the first few days and all values were above the high normal range even on the seventh day. A falling off of the vitamin A values coincided with the loss of clinical manifestations and the disappearance of the dermatitis.

The patients had the typical manifestations of vitamin A toxicity[2] in its characteristic temporal sequence after ingesting fish liver curry. *Istiophorus gladius* is a large fish that can attain a length of 8 to 10 feet: it thus had a large liver. The liver contains variable amounts of fish oil—for example, it contains 5-7% of oil, and this oil may contain 5000 to 25 000 IU vitamin A/g. Its oil content appears to be higher than that of other large fish in Sri Lankan waters like tuna, skipjack, and shark.[3] Interestingly, the tunny fish liver (Britain) also has a very high vitamin A content of about 50 000 IU/g oil. The clinical findings in our patients are comparable to those found after ingesting the liver of polar bear or seal, which also contain large amounts of vitamin A.[4]—S RAMACHANDRAN, physician, P PREMACHANDRA, biochemist, and D K D SILVA, technician.



Serum vitamin A concentrations in four patients with toxicity. *Conversion: SI to conventional units*—1 μmol/l ≈ 29 μg/100 ml.

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